Nocardiosis is caused by the gram-positive, filamentous, partially acid-fast bacilli *Nocardia,* which includes more than 50 ubiquitous, slow growing aerobic species.[1] Approximately 500 to 1000 cases of nocardiosis occur in the United States per year.[2] Primary cutaneous nocardiosis, most often caused by *Nocardia brasiliensis*, typically affects immunocompetent individuals with a history of trauma and is grouped into three entities: lymphocutaneous infection, mycetoma, and superficial skin infection including ulceration, abscess, and cellulitis. *N asteroides* complex typically causes skin infection from hematogenous dissemination from a pulmonary focus and is frequently associated with systemic disease.[3] Treatment should include evaluation for systemic disease as well as a minimum of 6 weeks of antibiotic therapy with trimethoprim-sulfamethoxazole, the treatment of choice for nocardiosis; treatment may be needed for up to 1 year.

Given the difficulty in isolating *Nocardia* spp, it is crucial to notify the laboratory if nocardiosis is suspected so that the appropriate time (3 to 21 days) and growth conditions are provided.[1,3,4] As demonstrated in this case, it is imperative for the clinician to follow up on abnormal or unexpected results, and to contact the laboratory and other involved physicians. Dermatologists must be aware of this potentially difficult to diagnose organism as a potential cause of nonhealing ulceration and other cutaneous infections.

James M. Graham, MD,[a] Kyle B. Bartlett, BS,[b] Annie Gonzalez, MD,[a] Jane L. Messina, MD,[a] and Philip D. Shenefelt, MD[a]

Department of Dermatology and Cutaneous Surgery, University of South Florida Morsani College of Medicine,[a] Tampa, and University of Miami Miller School of Medicine,[b] Florida

Funding sources: None.

Conflicts of interest: None declared.

Correspondence to: James M. Graham, MD, Department of Dermatology and Cutaneous Surgery, USF Morsani College of Medicine, 13330 USF Laurel Dr, 6th Floor, Tampa, FL 33612

E-mail: Jgraham1@health.usf.edu

REFERENCES

1. Young LS, Rubin RH. Mycobacterial and nocardial diseases. In: Rubin RH, Young LS, editors. Clinical approach to Infection in the compromised host. 4th ed. New York: Kluwer Academic/Plenum; 2002. pp. 257-60.
2. Centers for Disease Control and Prevention. Available from: http://www.cdc.gov/nocardiosis/infection.
3. Outhred AC, Watts MR, Chen SC, Sorrell TC. Nocardia infections of the face and neck. Curr Infect Dis Rep 2011;13:132-40.
4. Greenfield RA. Nocardiosis. Medscape. Available from: http://emedicine.medscape.com/article/224123-overview.

http://dx.doi.org/10.1016/j.jaad.2014.02.047

Giant cellulitis-like Sweet syndrome in the setting of autoimmune disease

To the Editor: An unusual presentation of Sweet syndrome, giant cellulitis-like type, has recently been reported in the setting of malignancy and morbid obesity.[1] We report a case of giant cellulitis-like Sweet syndrome in a patient with autoimmune disease in the absence of malignancy.

A 54-year-old woman with history of sicca syndrome, recurrent primary biliary cirrhosis (PBC) despite a liver transplant for PBC 16 years previously, and morbid obesity presented with a new red rash of at least 3 days' duration. She was initially hospitalized for *Escherichia coli* bacteremia (1 month before rash onset) that was treated with intravenous cefepime for 2 weeks; subsequent bacterial blood cultures were negative. Despite successful eradication of the bacteremia, chills and fevers associated with the new rash developed. Initially the rash was treated as cellulitis but did not respond to treatment with intravenous vancomycin.

On examination (Fig 1), there were multiple large, scattered, erythematous, edematous, indurated papules and confluent plaques on her buttocks, extremities, and trunk; the eruption progressed to involve the head and neck. Punch biopsies of a papule and plaque on the patient's back revealed prominent papillary dermal edema with a mixed inflammatory cell infiltrate composed of abundant neutrophils intermixed with lymphocytes, histiocytes, and eosinophils (Fig 2), consistent with Sweet syndrome. Leukocytoclastic vasculitis was not noted. Serum blood counts and chemistry demonstrated a normocytic anemia with hemoglobin of 8.1 g/dL, normal white blood count of 4.1 K/μL (with a slightly increased neutrophil count of 77%), and increased creatinine of 1.7 mg/dL. Tissue cultures for bacteria and fungi were negative. The patient was treated with a prednisone taper over a course of 40 days. The lesions improved rapidly within 48 hours after initiation of treatment. Rare clinical presentations of Sweet syndrome include necrotizing fasciitis and cellulitic types,[1-5] and do not respond to antibiotics.[3] Widespread "giant cellulitis-like" Sweet syndrome, such as presented in this case, was recently reported in 3 patients.[1] All of the cases described obese patients with recurrent fever and widespread infiltrated

Open access under CC BY-NC-ND license.

Fig 1. Giant cellulitis-like Sweet syndrome. Initial presentation with erythematous, edematous, indurated papules and confluent plaques on the right back (superior) and buttock (inferior; intergluteal cleft marked by *arrow*).

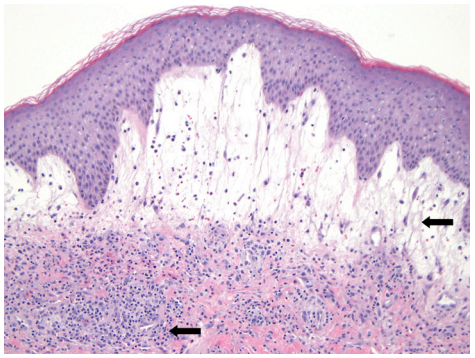

Fig 2. Giant cellulitis-like Sweet syndrome pathology. Prominent papillary dermal edema (top *arrow*) with abundant dermal neutrophils (bottom *arrow*).

plaques with bullous characteristics. Histopathology revealed classic findings of Sweet syndrome. Two patients were found to have an underlying malignancy (multiple myeloma or breast cancer); the other patient had no identified malignancy or autoimmune disease.

To our knowledge, an extensive cellulitic presentation of Sweet syndrome has only been described in the 3 patients mentioned here. Like in these cases, our patient had severe widespread eruption of giant cellulitis-like plaques and was morbidly obese. However, to date, no malignancies have been identified in our patient. Because Sweet syndrome may herald onset or recurrence of malignancy,[1,3] routine screenings and close follow-up are recommended. Our patient had classic Sweet syndrome in the setting of precedent bacteremia and inflammatory diseases (sicca syndrome and PBC), yet she presented with unusual giant cellulitis-like features. It is likely that giant cellulitis-like Sweet syndrome may be an uncommon clinical presentation for the different variants of Sweet syndrome and may not be restricted to the malignancy type.

Edidiong Celestine Ntuen Kaminska, MD, MBS,[a] *Adaobi I. Nwaneshiudu, MD, PhD,*[b] *Arlene Ruiz de Luzuriaga, MD, MPH,*[b] *Maria Tsoukas, MD, PhD,*[b] *and Diana Bolotin, MD, PhD*[b]

Division of Dermatology, Department of Medicine, NorthShore University HealthSystem,[a] *Skokie, Illinois, and Section of Dermatology, Department of Medicine, University of Chicago Medical Center,*[b] *Chicago, Illinois*

Funding sources: None.

Conflicts of interest: None declared.

Correspondence to: Edidiong Celestine Ntuen Kaminska, MD, MBS, Physician, Division of Dermatology, Department of Medicine, NorthShore University HealthSystem, 9933 Woods Drive, Skokie, IL 60077

E-mail: ekaminska@northshore.org

REFERENCES

1. Surovy AM, Pelivani N, Hegyi I, Buettiker U, Beltraminelli H, Borradori L. Giant cellulitis-like Sweet syndrome, a new variant of neutrophilic dermatosis. JAMA Dermatol 2013;149:79-83.
2. Kroshinsky D, Alloo A, Rothschild B, Cummins J, Tan J, Montecino R, et al. Necrotizing Sweet syndrome: a new variant of neutrophilic dermatosis mimicking necrotizing fasciitis. J Am Acad Dermatol 2012;67:945-54.
3. Withrow RA, Shenenberger D, Kalish VB, Krivda S. Cellulitis unresponsive to antibiotics. Sweet's syndrome. J Fam Pract 2011;60:149-51.
4. Dinh H, Murugasu A, Gin D. Sweet's syndrome associated with cellulitis. Australas J Dermatol 2007;48:105-9.
5. Crum NF, Higginbottom PA, Fehl FC, Graham BS. Sweet's syndrome masquerading as facial cellulitis. Cutis 2003;71:469-72.

http://dx.doi.org/10.1016/j.jaad.2014.03.025

Cutaneous polyarteritis nodosa presenting as a solitary blue toe

To the Editor: A woman in her 30s with a history of Crohn disease, quiescent on mesalamine, presented with a 1-year history of mild purple discoloration of the left great toe, which progressed to painful, burning nodules limiting movement of the digit. Her symptoms did not fluctuate with changing